Scheuermann's Disease: A Review

Scheuermann's disease (or more correctly, spinal osteochondrosis) is not confined to the thoracic spine: all parts of the spine may be affected. Major signs are endplate irregularity, more than 5° vertebral wedging, and sagittal overgrowth. Minor signs are Schmorl's nodes, anterior flattening of the vertebral endplate, and anterior detachment of a ring apophysis. Symptoms are of little diagnostic value. Scheuermann's affects 20 per cent to 30 per cent of the population; males and females equally.

A hereditary predisposition is a major factor in the aetiology of Scheuermann's disease. There is evidence that this condition follows an autosomal dominant pattern of inheritance.

Treatment consists of either exercises; bracing and exercises; or in very severe cases, surgery.

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This review summarizes current knowledge regarding the diagnosis, incidence, sex ratio, pathology, pathogenesis, aetiology and treatment of Scheuermann's disease.

Scheuermann's disease has been referred to by a number of titles (juvenile kyphosis, adolescent kyphosis, apprentice's kyphosis, Scheuermann's juvenile kyphosis, osteochondritis deformans juvenilis dorsi, vertebral osteochondritis and spinal or vertebral osteochondrosis); of these probably the most accurate is spinal osteochondrosis (Stoddard and Osborn 1979). However in order to provide continuity with the literature, the commonly accepted label, Scheuermann's disease (SD), will be used here.

Diagnostic Criteria

Scheuermann's concept of this condition was that of 'an abnormal arcuate and fixed kyphosis developing around puberty and caused by wedge-shaped deformity of one or more vertebrae which show certain radiographic peculiarities' (Sorensen 1964 p.18). Owing to the variability of the symptoms of SD, clinical symptomatology is not an acceptable method of defining the presence of this condition, and a number of radiological features have come to be accepted as indicative of SD.

Butler (1955) described these as follows: wedge shaped vertebral bodies (VB), increased anteroposterior diameter of the VB, irregular shaped and narrowed disc spaces, kyphosis or loss of lordosis, Schmorl's nodes, a flattened area on the superior surface of the VB in the region of the epiphysial ring anteriorly, and a detached epiphysial ring anteriorly. He has also pointed out that in the milder or localised forms of SD there may not be any deformity, and that all parts of the spine may be affected (Butler 1961). Butler's criteria were slightly modified and used by Taylor *et al* (1988) in their evaluation of SD as an aetiological factor in lumbar disc prolapse. They divided their criteria into those they considered to be major: endplate irregularity, wedging (more than 5°), and sagittal overgrowth (increased anteroposterior diameter of the VB), and those they considered minor: Schmorl's nodes, anterior flattening of the vertebral endplate (a lesser degree of wedging), and anterior detachment of a ring apophysis.

Incidence

Estimates of the incidence of SD have been based on clinical criteria, a combination of clinical and radiological criteria, and on radiological criteria alone. This variation in method of diagnosis, in addition to a variation in the criteria used for each of these methods, is chiefly responsible for the large discrepancy between authors on the incidence of SD (see Table 1).

Although the variation in the literature of the incidence of SD is large, it is not as large as Fisk *et al* (1982) purported it to be. These authors wrote that the recorded incidence of SD varied from 0.4% to 88%. The only occurrence of the figure 88% in the literature is Scheuermann's report that 88% of his patients with SD were boys

Table 1: Incidence of Scheuermann's disease*

Author	*Year*	*Number*	*Sex*	*Clin*	*X-Ray*	*Clin & X-Ray*	*Comments*
Hinricsson	1943	4465	M	8.2%	—	—	
Wassmann	1951	1500	M	—	—	3.2%	Rural
Wassmann	1951	750	M	—	—	0.4%	Urban
Dameron & Gulledge	1953	1000	M	8.3%	6.1%	—	
Hult	1954	91	M	13%	28%	—	
Bonne	1955	77246	M	—	—	0.42%	Rejected/ partially fit
Alexander	1977	400	M	—	31%	—	Unselected
Alexander	1977		F	—	21%	—	Unselected
Stoddard & Osborn	1979	853	M&F	—	13.1%	—	Rural
Stoddard & Osborn	1979	925	M&F	—	42.6%	—	Urban-backache
Fisk	1982	96	M	—	—	60%	
Fisk	1982		F	—	—	23%	

*Modified after Sorensen (1964).

(Scheuermann 1921). With the exception of the 88% mentioned by Fisk *et al* (1982), their own estimate of 60% for the incidence of SD for males is the only figure above 31% in the literature (Stoddard and Osborn's 42.6% refers to the percentage of backache sufferers with SD, amongst whom the incidence of SD was found to be twice as great as in the general population (Stoddard and Osborn 1979)). Based on the data from Alexander (1977) and Fisk *et al* (1982), who used radiological criteria similar to those advocated by Butler (1955) and Taylor *et al* (1988), and excluding Fisk's unusually high figure of 60% for males, the incidence of people affected by SD in the general population is estimated as being between 20% and 30%.

Sex Ratio

Scheuermann (1921) considered that SD almost exclusively affected boys, and Wassmann (1951) cited Holund's study, based on Scheuermann's patients, to support his conclusion that SD chiefly affects males. Nevertheless, a number of authors (Table 2) have found either no appreciable difference in the sex ratio or that a greater number of females than males were affected in a series of patients with SD. Bradford *et al* (1974) found that in their patients with SD (n = 168), the female-to-male ratio was two to one.

For all 1,338 cases reviewed by Sorensen (1964), the average sex ratio was 58% males compared with 42% females. Sorensen concluded that there was no distinct sex difference. This opinion was shared by Bradford *et al* (1974).

Pathology

Bradford and Moe (1975) obtained specimens for histological examination from two patients with active SD. The specimens were removed during anterior spinal fusions performed to correct the deformity. Macroscopically they found that the endplates were irregular and disrupted by protrusions of disc material into the bony spongiosum of the VB (Schmorl's nodes). Histological examination of the endplate, VB and disc failed to reveal any abnormality.

Aufdermaur (1981) examined 100 spines between 8 and 20 years of age. In five of these spines he found changes similar to those described by Bradford and Moe (1975), however, unlike these authors, Aufdermaur considered that the endplate was not normal. Specifically he found fibre defects and also areas completely devoid of collagen fibres. Furthermore, although Bradford and Moe found no ultrastructural abnormality of the endplate, VB or disc, an electron microscopic study by Aufdermaur and Spycher (1986) revealed irregularly arranged collagen fibrils within the areas described by other authors as Schmorl's nodes.

Pathogenesis

A number of theories have been proposed as to the mechanism leading to vertebral wedging, considered to be pathognomonic of SD. These theories must also attempt to explain endplate irregularity which is found in up to 95% of patients with SD (Bradford *et al* 1974).

A likely explanation for the disruption of the endplate is a congenital defect allowing disc material to protrude into the VB. Schmorl (Schmorl and Junghanns 1971) believed that SD occurred chiefly in those persons who had congenital indentations of the discs in the region of the nucleus pulposus (NP). He reported that in the region of these indentations the endplate was thinner than normal and that this created a region of decreased resistance. He believed that the stress of physical labour or sport during early adolescence could then cause fissuring of the endplate with resulting prolapse of the NP into the spongiosa of the VB. The growth zone (consisting of hyaline cartilage directed towards the bony surface) was then extensively destroyed and the loss of both elasticity and tissue of the NP brought the VB closer together. As the facet joints blocked the VB posteriorly, only the anterior edges

Table 2:
Sex ratio in patients with Scheuermann's disease†

Author	*Year*	*Pts*	*M*	*F*	*Remarks*
Scheuermann	1920	105	88%	12%	Mostly adolescents
Scheuermann	1936a	202	82%	18%	Adolescents & adults
Albanese	1936	150	33%	67%	All 10-20 years old
Schildbach	1937	167	43%	57%	Mostly adolescents
Dittmar	1939	88	64%	36%	Adolescents & adults
Lotze	1939	51	49%	51%	47 under 19 years
Nathan & Kuhns	1940	75	33%	67%	Mostly adolescents
Hodgen & Frantz	1941	50	22%	78%	Mostly adolescents
Holund	1943	220	72%	28%	Adolescents & adults
MacGowan	1944	130	49%	51%	Mostly adolescents
Burdzic & Wuensch	1954	100	49%	51%	76 under 18 years
		1338	58%	42%	
Present series					
Probands		103	48%	42%	
M.A. (Aarhus)		93	77%	23%)	All patients under
M.B. (Copenhagen)		146	81%	19%)	20 years

†Sorenson (1964)

of the VB moved towards each other. The pressure of the anterior VB on each other, and the resulting damage to the growth plate, then led to wedging.

Schmorl considered 'ossification gaps' in the endplate to be an important site for herniation of disc material. These gaps resulted from incomplete closure of the endplate at the site of previous notochord penetration. Investigating cases of SD Laederer found 'hernias of the core of the NP at these sites' (Schmorl and Junghanns 1971 p.348). Laederer believed that ossification gaps ('abortive ossification centres') were a more important cause of Schmorl's nodes than the perforations caused by the passage of blood vessels through the endplate during development (Schmorl and Junghanns 1971). Aufdermaur (1981) disagreed with Schmorl and Laederer as to the importance of ossification gaps, and pointed out that these gaps occurred with equal frequency in normal spines. He also considered vascular channels or vascular scars to be unlikely sites for Schmorl's nodes and was unable to find disc tissue within either of these areas.

It is likely that the cause of anterior detachment of the ring apophysis is also related to disc protrusion. Butler believed that the detachment of the apophysial ring anteriorly was caused by the pressure of extruded disc material passing under the apophysial ring. He considered that disc material passed through the endplate and then moved forward towards the periphery 'beneath the bony centres of the marginal ring epiphysis' (Butler 1961 p.763).

Aetiology

Many authors were of the opinion that SD was a familial condition however the specific mode of inheritance was not clear (Halal *et al* 1978).

Although Sorensen (1964) was unable to demonstrate any common aetiology in patients with SD, he did find a high familial incidence of this condition. Bradford *et al* (1974) found that 25% of cases with SD had a positive family history.

Several authors have attempted to identify the specific mode of inheritance in SD. Kewalramani *et al* (1976) present evidence that suggests that the same genetic defect is responsible for both Charcot-Marie-Tooth syndrome (CMTS) and SD. Their findings are based on detailed clinical and laboratory studies of a family with three cases of SD and CMTS in three generations. The family under examination showed an autosomal dominant mode of inheritance. Further evidence of autosomal dominant inheritance was found by Halal *et al* (1978).

The role of strenuous physical activity, whether the result of occupation, sport or other activity, has been emphasised by many authors considering the aetiology of SD (Sorensen 1964). Scheuermann (1921) specified strenuous physical work as an important aetiological factor and found that the majority of those with SD were farmworkers. In contrast, Stoddard and Osborn (1979), in their study of the relationship of SD to spondylosis, found that rural workers had a far lower incidence of SD than subjects from urban areas. Admittedly the urban subjects all had prior complaints of backache. Nevertheless, one would also expect a significant proportion of miners and farmworkers to suffer from backache.

Sorensen (1964) found that only a small proportion of patients with SD had done physical work.

Conservative Treatment

Physiotherapy

Because of the variation in signs and symptoms between patients with SD, a careful subjective and objective examination is needed to pinpoint problems specific to a particular individual.

Considerable improvement of the kyphosis is possible with exercises alone, however successful conservative correction of the deformity is only possible in the skeletally immature patient (Beyeler *et al* 1979). Closure of the iliac apophyses (at the time of initial treatment) is a useful guideline to skeletal maturity, however it is still possible to

obtain correction so long as the vertebral ring apophyses have not closed (Bradford 1977).

The following exercises have been found to be effective for correction of the deformity: pelvic tilting to decrease lumbar lordosis, muscle stretching to overcome contractures (chiefly pectorals and hamstrings), and thoracic extension to strengthen the thoracic extensor muscle groups (Bradford 1981). Posture correction is also important and simple instructions emphasizing standing as tall as possible, such as those suggested by McKenzie on page 35 of his book *Treat Your Own Back* (1985) are recommended. The parents of young patients will also need to be instructed in the exercise programme (Beyeler *et al* 1979), and consideration will need to be given to the special problems associated with adolescence and puberty (Gallagher and Harris 1976).

Although back pain in adolescents with SD is held to be uncommon (Kling and Hensinger 1984), Bradford *et al* (1974) admitted that poor questioning and early treatment contributed to the low incidence of back pain (15%) that they reported in their series of young patients with SD. It is thought to be higher (50% or more) in mature patients with SD (Bradford 1977).

The main advantage of first using exercises in an attempt to relieve pain, is that the patient's attention is focused on an exercise programme which is inherently beneficial (see above). There is a strong possibilty that the endorphin-mediated analgesia system contributes to any reduction in pain following exercise (Fields 1982).

The avoidance of specific activities that cause pain, combined with a comprehensive back care programme, are self-evident methods of achieving pain reduction. There are a number of back care programmes designed for the patient's own use. One such programme suitable for the patient with SD (with the exception of the situp exercise) is contained in a book by Professor White (1983) entitled *Your Aching Back.*

Consideration of the pathology of SD suggests that mobilisations may be of little benefit in the treatment of the primary pathology. Nevertheless gentle mobilisations may be beneficial in the treatment of patients with backache secondary to the presence of SD (Stoddard 1983). Contemplation of more vigorous treatment or activity, such as manipulation or patient participation in contact sport, must involve consideration of the possibility of cord compression or ischaemia. Ryan and Taylor (1982) discuss three patients with SD and a sharp kyphosis in one of whom relatively minor trauma was sufficient to cause neurological compromise. They point out the importance of factors such as local anatomical variation and the rate of change of the kyphotic angle, which may predispose to the onset of cord compromise. Although this form of spinal cord compression is rare (Bradford 1977, Ryan and Taylor 1982) the vulnerability of patients with severe SD to trauma or a sudden increase in the kyphotic angle, particularly if the deformity is sharply angular rather than present over a large number of segments, suggests that both manipulation and patient participation in contact sport should be avoided. The decision as to whether or not an adolescent with SD should participate in other forms of sport, will depend to some extent on the effect this has on the patient's symptoms. However it should be borne in mind that some activities, such as rowing, are likely to make the condition worse (Endler *et al* 1980).

With regard to other forms of treatment such as electrotherapy, it has been found that several applications of continuous ultrasound (1.0 w/cm^2) followed by short wave diathermy, will dramatically decrease the pain associated with SD. Similarly, interferential (90-100 Hz) can be used to assist in the control of pain (unpublished data).

Bracing

The selection of patients for bracing in preference to exercises alone depends on the severity of the deformity (usually greater than 40°) and the type of vertebral changes apparent on x-ray (Bradford *et al* 1974). This selection may also be influenced by the views and temperament of the physician in charge (Beyeler *et al* 1979).

The patient is fitted for a Milwaukee brace or, more rarely, a Risser antigravity cast. The brace is worn full-time (16-22 hours a day) usually for a period of at least one or two years. Once correction is achieved (the kyphosis is less than 40° and wedging is approaching normal; 5° or less) weaning from the brace is begun. Any loss of correction results in the patient spending increased time in the brace. Exercises are done both in and out of the brace and are as described previously: active thoracic hyperextension, pelvic tilting and stretches (Bradford 1977). It is worth noting that an American trial of additional time out of the brace in order to permit patients to participate in competitive athletics did not result in their clinical deterioration (Benson *et al* 1977).

Operative Treatment

Surgery is rarely necessary. Indications include a severe structural kyphosis in a patient who has completed growth and who is unresponsive to conservative management; significant disabling pain; and neurological signs and symptoms secondary to the kyphosis (Bradford 1977). Most importantly a careful evaluation of the problem must be made: the natural history of the condition without treatment must be weighed up against the risk, expense and possible complications of surgery (Bradford 1981).

In order for surgery to be effective, particularly in the skeletally immature patient, a two stage procedure is necessary. Anterior spinal fusion is followed by a second operation to achieve posterior spinal fusion. A transthoracic approach is frequently used for the anterior spinal fusion. The anterior longitudinal ligament is divided, the apical

7 or 8 vertebral discs removed and the gap bridged with rib or iliac bone graft (Bradford 1977).

The second operation is performed as soon as the patient has recovered from the first (Ryan and Taylor 1982). The posterior fusion extends the length of the kyphosis and is usually achieved using Harrington compression rods. The patients is then mobilised in a Risser cast about ten days after the operation (Bradford 1977). The length of time in the cast, and the timing of the resumption of normal activities, are at the surgeon's discretion. Specific postoperative physiotherapy is usually unnecessary as the deformity has been corrected independently of muscle tone.

Conclusions

A number of miconceptions have arisen concerning SD and the following points should be noted:

- SD is not confined to the thoracic spine. Any part of the spine may be affected, however descriptions of osteochondrosis of the cervical spine are rare.
- Boys are neither more nor less likely than girls to have SD.
- There is no good evidence that strenuous physical activity during adolescence is a significant factor in the aetiology of SD.
- SD is not rare. Between 20% and 30% of the general population are affected to some degree.
- Contrary to past opinion (Sorensen 1964), exercises are often beneficial in the treatment of both mild and moderate SD.

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